jnm / CASE REPORT

^{67}Ga LOCALIZATION IN ACUTE CEREBRAL INFARCTION

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Gallium-67 first gained recognition as a possible tumor-specific scanning agent in epithelial and reticuloendothelial neoplasms. Recent reports have described localization of this isotope in various nonmalignant disease states including acute inflammatory processes and chronic granulomatous diseases. A case is presented of ^{67}Ga-citrate localization in acute cerebral infarction.

Gallium-67 first gained popularity as a tumor-specific scanning agent following the published report by Edwards and Hayes in 1969 (*1*). The initial studies performed indicated increased uptake particularly in neoplasms of epithelial and reticuloendothelial origin. The most avid accumulation of isotope occurred in viable tumors and appeared decreased in fibrotic and necrotic neoplasms and in tumors manifesting response to radiation therapy and to chemotherapy.

More recently attention has been drawn to ^{67}Ga localization in various nonmalignant acute inflammatory and chronic granulomatous processes, particularly involving the lungs and liver (*2–5*).

CASE REPORT

BN is a 58-year-old black man who presented with a sudden onset of left hemiparesis, left homonymous hemianopsia, and left hemisensory loss. A brain scan was performed 2 weeks following the acute episode. This initial scan (Fig. 1) performed 1 hr after the intravenous administration of 15.0 mCi ^{99m}Tc-pertechnetate revealed the classic picture of a cerebral infarction involving branches of the right middle cerebral arterial group.

Electroencephalography revealed an abnormal tracing with excessive theta and delta activity in all right hemispheric leads, especially the right parasylvian region.

Immediately after the pertechnetate brain scan, 3.0 mCi of ^{67}Ga-citrate were administered intravenously, and the patient was scanned 72 hr later (Fig. 2). The scan pattern with ^{67}Ga was identical to that of the previous pertechnetate scan.

Carotid arteriography revealed complete occlusion of the right middle cerebral arterial group.

DISCUSSION AND CONCLUSION

There has been an expanding volume of reports in the recent literature concerning nontumor-specific ^{67}Ga localization in various nonmalignant disease states. Despite an increasing interest in ^{67}Ga as a scanning agent, little information has been obtained concerning its mechanism of localization. There is some evidence by microautoradiography that ^{67}Ga localizes in the cytoplasm of tumor cells bound to granules appearing to represent lysosome granules. Microautoradiography of inflammatory lesions suggest ^{67}Ga is distributed in the infiltrating neutrophilic leucocytes and is taken up by phagocytosis of histiocytes in active granulomatous lesions (*3*).

Recently, attention has been focused on the use of ^{67}Ga for the evaluation of intracranial neoplasms (*6,7*) with the possible potential use of this isotope in determining the presence of tumor recurrence following surgery and detecting underlying tumor in the presence of an associated craniotomy site.

The present case report suggests that the ultimate value of examination with ^{67}Ga for intracranial neoplasm may be limited, especially in the presence of associated acute ischemic tissue necrosis. A recent report indicates apparent ^{67}Ga localization in myocardial infarction (*8*) and further documents the

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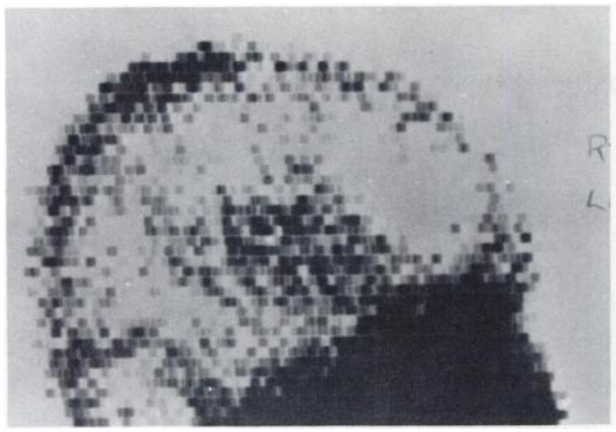

FIG. 1. Right lateral view of ^{99m}Tc-pertechnetate brain scan showing abnormal isotope localization in region supplied by right middle cerebral artery.

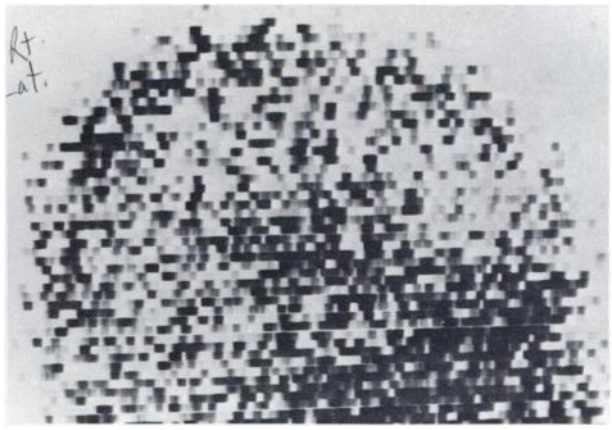

FIG. 2. Right lateral view of ^{67}Ga-citrate brain scan showing isotope localization pattern similar to that seen with ^{99m}Tc-pertechnetate.

nonspecificity of localization of this isotope in nonneoplastic disease states. It may be postulated that the mechanism of ^{67}Ga localization in cerebral infarction is similar to that manifested with inflammatory lesions.

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